Journal of Abnormal Psychology
2000, Vol. 109, No. 1, 26–39

0021-843X/00/$5.00 DOI: 10.1037//0021-843X.109.1.26

Emotional Processing in Posttraumatic Stress Disorder

Brett T. Litz
Boston Department of Veterans Affairs Medical Center and
Boston University School of Medicine

Susan M. Orsillo
Oklahoma State University

Danny Kaloupek
Boston Department of Veterans Affairs Medical Center and
Boston University School of Medicine

Frank Weathers
Auburn University

The emotional deficits associated with posttraumatic stress disorder (PTSD) are the least understood and the most understudied aspect of the syndrome. In this study, the connection was evaluated between trauma–context reactivity and subsequent emotional deficits in PTSD. Combat veterans with PTSD and well-adjusted veteran control participants were exposed to reminders of combat, after which their emotional behavior was assessed in response to a series of emotionally evocative images. Under the neutral condition, both groups exhibited emotional behavior modulated by stimulus valence. Partially consistent with the conceptual model described by B. Litz (1992), the PTSD group exhibited suppressed expressive–motor responses to positively valenced images, in comparison with the control group, only after being exposed to a trauma-related prime. Contrary to expectations, the PTSD group showed no augmentation of emotional response to negatively valenced cues after being exposed to trauma reminders. However, the PTSD group responded to all images, in both prime conditions, with higher heart rate reactivity, suggesting an automatic preparation for demand or threat in any uncertain emotional context. Possible causes and consequences of these results are discussed.

Disrupted emotional experiences are central features of the long-term psychological consequences of traumatic events. Individuals with posttraumatic stress disorder (PTSD) often report two different types of problems related to emotion. On the one hand, patients with PTSD report intense negative emotional reactions when reminded of their trauma (e.g., fear, sadness). On the other hand, individuals with chronic PTSD report disinterest in circumstances that would otherwise elicit emotion and a lack of ability to experience and express emotions, the combination of which is referred to as *emotional numbing* (American Psychiatric Association; [APA], 1994). Several theorists have argued that these two types of experiences are reciprocal or phasic and that their interplay is fundamental to the nature of PTSD (Horowitz, 1986; van der Kolk, 1987; van der Kolk, Greenberg, Boyd, & Krystal, 1985). However, the mechanism by which states of cued negative emotional reactivity alternate with episodes of emotional unresponsiveness has not been explicated sufficiently. Also, there is a good deal of confusion in the clinical literature about whether emotional-numbing-related problems are a phasic response pattern or, rather, coincident with reexperiencing and hyperreactivity symptoms. Although there has been considerable empirical research conducted on negative emotional responses to various challenge tasks in PTSD (e.g., Pitman, Orr, Forgue, de Jong, & Claiborn, 1987), there has been no systematic research on emotional response deficits in PTSD and no experimental investigations of the relationship between these two response classes (Davidson & Foa, 1991).

In this study, we examined the effects of trauma-cue exposure on subsequent emotional behavior in PTSD. In our view, PTSD patients are not generally numb and unresponsive, but rather what has previously been labeled as emotional numbing is best characterized as a deficit in emotional processing arising from episodes of hyperemotionality brought on by exposure to trauma cues (Litz, 1992). When in a state of acute distress brought on by reminders of their trauma, patients with PTSD are hypothesized to experience less intense positive feelings and to be more reactive to negative cues. Before outlining the specifics of the model that generates these predictions, we review briefly the empirical literature on two classes of emotional problems in trauma and summarize the alternative theories of emotion in PTSD.

Researchers of trauma have made considerable strides in understanding emotional responses to trauma-related cues in PTSD. This is due, in part, to the clear operational definition of these problems in the nosology and the extensive history of measuring fearful and anxious behavior in other anxiety disorders. Investigators have shown that patients with PTSD react intensely to cues reminiscent

Brett T. Litz and Danny Kaloupek, National Center for PTSD, Boston Department of Veterans Affairs Medical Center and Department of Psychiatry, Boston University School of Medicine; Susan M. Orsillo, Department of Psychology, Oklahoma State University; Frank Weathers, Department of Psychology, Auburn University.

This research was conducted at the National Center for PTSD, Behavioral Science Division at the Boston Department of Veterans Affairs Medical Center and was supported by a Merit Review Grant from the Department of Veterans Affairs. We thank Sherry Beaudreau for her tireless work in support of this research. We also thank Eric Bergman for his assistance.

Correspondence concerning this article should be addressed to Brett T. Litz, National Center for PTSD (116B-2), Boston Department of Veterans Affairs Medical Center, 150 South Huntington Avenue, Boston, Massachusetts 02130. Electronic mail may be sent to brett.litz@med.va.gov.

of their trauma. Such patients often exhibit an integrated negative emotional response in trauma-related contexts, involving central and peripheral nervous system activity, and a variety of unpleasant feelings (Malloy, Fairbank, & Keane, 1983; Pallmeyer, Blanchard, & Kolb, 1986; Pitman et al., 1987; Rauch, van der Kolk, Fisler, & Alpert, 1996).

A major obstacle to the study of emotional response deficits in PTSD is that the concept of emotional numbing is ambiguous and poorly operationally defined. For example, what is meant by *restricted range of affect?* On its face, this symptom suggests a generalized limitation in the capacity to feel and express emotions. Such a broad definition is untenable given the intense emotional responses associated with reliving experiences in trauma. If PTSD patients are restricted in some of their emotional responses, which emotions are constricted? Furthermore, are the putative restrictions in affect a problem in expressive behavior, felt emotion, or both? If the problems with numbing are phasic, as some researchers suggest, what triggers the activation of this process? A critical examination of the parameters of emotional-processing deficits in PTSD is needed to clarify these issues.

Several theories have been proposed to explain why PTSD patients develop restrictions of their emotional experience. First, the behavioral formulation of PTSD suggests that emotional numbing is a consequence of the chronic avoidance of trauma reminders and reactions (e.g., Keane, Fairbank, Caddell, Zimering, & Bender, 1985). Findings from several studies reveal that an avoidance explanation of emotional numbing is not sufficient. One factor analytic study revealed that reports of emotional numbing were distinct from avoidance symptoms (e.g., Foa, Riggs, & Gershuny, 1995). In addition, avoidance symptoms account for a negligible proportion of the variance in reports of the three emotional numbing symptoms (disinterest, detachment, and restricted range of affect) after other clusters of PTSD symptoms are taken into account (Litz et al., 1997).

Second, several investigators have proposed deficits of emotion in PTSD to be analogous to the sequelae observed in infrahumans exposed to inescapable shock, most notably catecholamine depletion (e.g., van der Kolk et al., 1985), and conditioned analgesia (Pitman, van der Kolk, Orr, & Greenberg, 1990). Foa and her colleagues argued cogently that emotional numbing is a variation of the conditioned analgesia seen in infrahumans exposed to uncontrollable and unpredictable aversive stimulation (Foa, Zinbarg, & Rothbaum, 1992). These researchers suggested that emotional numbing is a phasic response cued by trauma-related contexts, akin to cued states of stress-induced analgesia brought on by exposure to contexts in which uncontrollable shock was administered. This model has considerable conceptual appeal, especially in its attempt to explain how numbing-related phenomena are cued by trauma-related contexts. It also leads to testable empirical propositions. For example, the model predicts conditioned analgesia to be cued by trauma-related stress responses. What can be inferred also is that outside of trauma-related contexts, analgesic phenomena (numbing) are quiescent. However, there has been little experimental research attempting to validate the model in humans, although there is some evidence of conditioned analgesia after a trauma-related challenge in PTSD (Pitman et al., 1990). In addition, the relationship between conditioned analgesia and emotional behavior in traumatized humans remains unspecified.

Third, information-processing theories have been developed to explain the emotional consequences of trauma. Horowitz (1986) proposed that trauma creates two opposing sets of internal processes, intrusion and denial, that individuals use to cope with and resolve responses to extreme stressors. The intrusion phase of adjustment includes the hallmark cognitive and emotional symptoms of PTSD: painful reexperiencing and hyperemotionality. Such intrusive experiences trigger an opponent process of ideational and emotional denial that represents the defensive phase of adjustment. The function of denial is to ward off painful affects and memories related to the trauma. Emotional numbing is a component of denial that allows patients with PTSD to minimize the feelings associated with traumatic memories. A traumatized person is said to shift back and forth between generalized unresponsiveness (numbing) and intrusion until resolution of the trauma occurs. Although Horowitz's model has had widespread heuristic value, there have been few accounts of its application in the study of chronic forms of PTSD. Furthermore, there is no evidence that emotional numbing symptoms occur generally as a phasic response after a period predominated by intrusive symptoms (e.g., Joseph, Yule, & Williams, 1995).

A number of investigators have expanded on Horowitz's information-processing model, incorporating constructs from theories of social cognition, cognitive science, and Lang's (1985) bio-informational model of fear, to explain the mechanisms that cause PTSD symptoms (Chemtob, Roitblat, Hamada, Carlson, & Twentyman, 1988; Foa, Steketee, & Rothbaum, 1989; Litz & Keane, 1989). A basic tenet of these models is that cues reminiscent of the trauma trigger a neural network of trauma-related associations and action potentials that create conditioned emotional responses and reexperiencing phenomena. PTSD is distinguished from other disorders by the unusually coherent and stable network of trauma memories that requires few matching elements in the environment before the network is activated (e.g., Foa et al., 1989). These information-processing models have accounted effectively for the re-experiencing and hyperemotionality symptoms of PTSD, but have not proposed specific limiting conditions for the emotional deficits.

Litz (1992) invoked Leventhal's (1984) perceptual–motor theory of emotion and synthesized various tenets of the information-processing models of PTSD to explain deficits of emotional processing in PTSD. In this perspective, unlearned expressive–motor programs, which are the building blocks to all emotional experience (Leventhal, 1984), are not affected by stress, trauma, or posttraumatic pathology. The expressive–motor functions that were available to the individual before they were traumatized are intact, as are pretraumatic, elaborated emotional knowledge or schemas. Thus, Litz argued that the capacity to experience and express a variety of emotions is unaltered in PTSD, and the construct of emotional numbing fails to take into account the complex, dimensional, and context-dependent nature of emotional behavior in traumatized individuals.

Litz (1992) argued that there is a primacy of conditioned aversive emotional reactivity and associated re-experiencing problems in posttraumatic adjustment. Because the network of trauma memories is broadly generalized across the domains of thought, image, action, and feeling, it is more readily triggered and, therefore, more accessible to experience than are other emotion networks (Chemtob et al., 1988). When the trauma network is activated, other, more adaptive responses to interpersonal stimuli are less accessible and less likely to influence behavior (e.g., McNally, Litz, Prassas, Shin, & Weathers, 1994). Expressive behaviors associated with moderate to high pleasant feeling are expected to be less accessible to experience when PTSD patients are in states of reexperiencing, characterized by high arousal and aversive emotion cued by trauma-related contexts. In addition, when patients with PTSD are in symptomatic, hyperemotional states, they should be more responsive to emotional contexts or cues that are consistent with that state (Litz, 1992). Thus, like the Foa et al. (1992) model, Litz's reformulation proposed that emotional-processing deficits in PTSD are context dependent. However, unlike the Foa et al. model, Litz's model specified differential emotional deficits associated with exposure to reminders of trauma.

The Present Study

In order to understand better the link between conditioned hyperemotionality and subsequent emotional-processing deficits in PTSD, we exposed two groups of Vietnam combat veterans (with and without PTSD) to an evocative trauma-related priming video (and to a neutral comparison video). Directly after presentation of the priming events, we assessed participants' emotional reactions to a set of photographic images that varied in hedonic valence (positive, neutral, and negative). Emotional responses were evaluated across a number of dimensions, including peripheral autonomic activity, expressive–motor responses, and self-reported emotional reactions.

We predicted a state-specific differential emotional-processing deficit to be characteristic of participants with PTSD. Specifically, patients with PTSD were expected to show a suppressed emotional response to positively valenced stimuli and an augmented response to negatively valenced images only after they were administered the trauma challenge. As Litz's (1992) model does not make specific predictions about the locus of emotional-processing deficits in PTSD, we were particularly interested in examining the relative effects of the priming manipulation on endorsements of emotional experience and expressive behavior. We also examined the effects of the priming manipulation on the concordance between participants' appraisal of their emotional state and expressive–motor reactions. If expressive behavior and evaluations or judgments of affective state are not linked, as they should be, this can reflect problems in emotional expressivity.

Method

Participants

Participants were 61, right-handed, male, Vietnam war-zone-exposed veterans recruited through hospital referrals, flyers, and newspaper advertisements. Participants were assigned to one of two groups on the basis of a clinical interview: those meeting diagnostic criteria for current PTSD (PTSD, $N = 32$) and those free of any current Axis-I psychiatric disorder (well-adjusted veterans [WAV], $N = 29$). The PTSD diagnosis was based on symptoms specified in the 4th edition of the *Diagnostic and Statistical Manual of Mental Disorders* (*DSM–IV;* APA, 1994), endorsed with an intensity of 1 or greater, and a frequency of 2 or greater within the last month, defined by the Clinician Administered PTSD Scale (CAPS; frequency and intensity values range from 0–4; Blake et al., 1990; see Weathers & Litz, 1994).

Design

The design was a 2 × 2 × 3 mixed factorial with diagnostic group (PTSD vs. WAV) as a between-groups factor and prime event (combat vs. neutral) and emotional valence category of photographic stimuli (positive, neutral, negative) as within-subjects factors. The order of presentation of the prime event was randomly assigned and counterbalanced. The photographic images were presented in a fixed block, randomized fashion.

Stimulus Materials

Priming event videos. The trauma prime videotape was composed of still photographic images of Vietnam combat situations and sounds from the war zone chosen to elicit negative emotional reactions in Vietnam combat veterans with PTSD. The neutral prime tape consisted of a series of still photographs of furniture set to background piano music. Each tape was 10 min in duration and was recorded and presented at the same sound level. Both priming tapes had been applied successfully previously in our laboratory (McNally et al., 1994; Weathers et al., 1996).

Emotionally valenced images. A total of 24 color photographic images were chosen from the International Affective Picture System (IAPS; Center for the Study of Emotion and Attention, National Institute of Mental Health [CSEA-NIMH], 1999). The IAPS is a large set of photographs validated extensively by Lang and his colleagues. We chose to examine emotional processing of photographic stimuli because this method has been used frequently in the study of normal human emotion (e.g., Cacioppo, Bush, & Tassinary, 1992). In addition, by using images from the IAPS, we were able to choose photographs with well-established emotion-eliciting properties (Bradley, Greenwald, Petry, & Lang, 1992; Greenwald, Cook, & Lang, 1989; Lang, Bradley, & Cuthbert, 1999). The IAPS images vary on two principal orthogonal dimensions of human emotion: valence (pleasantness) and arousal (degree of activation). Lang's research group has shown that participants' ratings of valence and arousal covary systematically with physiological reactions indicative of positive and negative affect states (Greenwald et al., 1989; Lang, Greenwald, Bradley, & Hamm, 1993). For example, zygomaticus major (zygomatic) activity (a facial muscle supporting the smile), measured electromyographically (EMG), is associated positively with valence judgments; corrugator activity (associated with a frown or scowl) is associated negatively with valence ratings.

On the basis of normative data provided by Lang et al., (1999), we chose an initial pool of IAPS images rated as highly pleasant, neutral (midscale), and very unpleasant. Images that contained obvious combat-related content were excluded. From this pool of images, we conducted a small-scale validation study to select our final set of stimuli. In all, 10 research assistants and colleagues, naïve to the experiment, rated the pool of IAPS images using our ratings procedure. On the basis of these ratings, we chose eight images that fell into the high, middle, and low tertiles. We also made sure that mean arousal ratings did not differ between the positive and negative categories. Table 1 shows the list of images chosen and the respective mean valence and arousal ratings from the validation study.

Apparatus and Response Measurement

The experiment was conducted in a sound-attenuated, temperature-controlled room. Participants were monitored using a video camera and intercom system. The priming video and IAPS images were presented via videotape. All stimulus events and ratings scales were presented on a 25-in. video monitor positioned 4 ft from the participant. Rating scales were presented by an IBM-compatible personal computer (PC), and participants made ratings via a mouse. Another PC was used to manage the collection and storage of physiological data. The display of rating scales and collection of psychophysiological data were controlled by audio signals recorded onto one channel of the videotape. The other audio channel was used to present instructions for the experiment and the sound track of the priming events via headphones.

Table 1
IAPS Images Used in the Study: Order of Presentation, Description, Emotional Valence Category, and Mean Valence and Arousal Ratings From the Validation Study

Description	Valence category	Valence	Arousal
Bloody male	Negative	2.33	4.19
Nude female	Positive	7.40	6.58
Building	Neutral	5.04	2.80
Baby	Positive	8.34	5.41
Rock climbing	Neutral	5.84	4.18
Male holding injured boy	Negative	1.60	6.49
Computer	Neutral	4.76	2.75
Sick male in hospital	Negative	2.26	6.55
Three babies	Positive	8.42	4.87
Three puppies	Positive	8.11	4.91
Cesarean scar	Negative	3.18	6.51
Freeway	Neutral	5.11	3.49
Battered female	Negative	2.57	5.80
Neutral male face	Neutral	5.27	3.96
Male with two kids	Positive	7.58	5.16
Dirty toilet	Negative	1.80	6.70
Roller coaster	Positive	7.32	7.34
Boy's face	Neutral	5.23	3.08
Male with beer	Neutral	5.26	4.08
Dying boy	Negative	1.80	6.69
Waterfall	Positive	7.73	6.88
Nude scene	Positive	7.51	7.18
Bloody, mangled hand	Negative	1.50	6.44
Basket	Neutral	4.47	3.36

Note. IAPS = International Affective Picture System.

Special software was written to present rating scales to participants. For emotion ratings, participants were instructed to select a number between 1 and 9 that represented their current level of valence (anchored from *unhappy/very unpleasant* to *happy/very pleasant*) and arousal (anchored from *calm* to *excited*). The ratings system was a Likert-type adaptation of the self-assessment manikin rating system used by Lang and his colleagues (e.g., Lang et al., 1993). Participants also completed a computer-generated version of the Positive Affect Negative Affect Schedule (PANAS; Watson, Clark, & Tellegen, 1988) at several points throughout the experiment.

We measured two indexes of peripheral autonomic activity, heart rate (HR) and skin conductance level (SCL), primarily to evaluate responsivity to the priming manipulation. Heart rate was recorded from 9-mm diameter SensorMedics Ag–AgCl electrodes (SensorMedics, Yorba Linda, CA) filled with Beckman electrolyte and attached by adhesive collars at standard lead I (arm) sites. Electrodes were connected to a Coulbourn Instruments high gain bioamplifier (S75-01, Coulbourn Instruments, Allentown, PA), and output from the amplifier was directed to a Coulbourn Instruments tachometer (S77-26) to yield a beats-per-minute (BPM) equivalent of each interbeat interval. Skin conductance was measured directly by a Coulbourn Instruments isolated skin conductance coupler (S71-23), using a constant 0.5-volt output, through 9-mm diameter SensorMedics Ag–AgCl electrodes. An isotonic paste was used as the conductive medium following the recommendations of Fowles et al. (1981), and electrodes were attached by adhesive collars to the hypothenar surface of the left hand.

Facial EMG recording was the principal method used to index expressive behavior. Surface EMG activity was measured over the zygomatic and the corrugator facial muscles. Expressive behavior in these two areas of the face are indicative of positive and negative affect states, respectively (see Cacioppo, Bush, & Tassinary, 1992). EMG activity was recorded via 4-mm diameter SensorMedics Ag–AgCl electrodes filled with Beckman electrolyte and attached by adhesive collars over the left corrugator and zygomatic sites, in accordance with recommendations of Fridlund and Cacioppo (1986). The EMG electrodes were connected to a Coulbourn Instruments high gain bioampifier (S75-01), which was set to filter any signal component less than 90 Hz or more than 1000 Hz. The signal was rectified and integrated with a time constant of 500 ms using a Coulbourn Instruments contour following integrator (S76-01).

All physiological signals were digitized by a Coulbourn Instruments Lablinc A/D Converter (L25-12) connected to a PC through a Coulbourn Instruments Lablinc Computer Interface (L18-16). We used Labtech Notebook software (Laboratory Technologies Corporation, Wilmington, MA) to automate sampling of physiological measures at a rate of 10 Hz and to convert values into appropriate measurement units (BPM for HR; microsiemens for SCL; microvolts for EMG).

Procedure

Overview. The study required three sessions. Session 1 was designed to collect diagnostic information and to introduce participants to the laboratory and establish an initial baseline for psychophysiological activity. Session 2, approximately 3 days after Session 1, was the first experimental session. It was followed approximately 1 week later by Session 3, the second experimental session. See Figure 1 for a schematic of the procedure.

Upon their initial arrival at the laboratory, for Session 1, participants were informed about the nature and requirements of the study and were asked to complete a consent form. Participants were then administered a diagnostic battery including the CAPS and the nonpatient (Axis-I) edition of the Structured Clinical Interview for *DSM-III-R* (Spitzer, Williams, Gibbons, & Faust, 1989). Interviewers were doctoral-level clinicians trained in the administration of both instruments using videotapes and formal didactic presentations. The results of each interview were reviewed by one of two investigators (BL or SO) before group assignment was made. On the first day, participants were also administered a series of self-report instruments designed to measure war-zone exposure (Combat Exposure Scale [CES], Keane et al., 1989), PTSD (Mississippi Scale for Combat-Related PTSD [M-PTSD], Keane, Caddell, & Taylor, 1988), comorbid symptoms (Beck Depression Inventory [BDI], Beck et al., 1961; Beck Anxiety Inventory [BAI], Beck, Epstein, Brown, & Steer, 1988), and mood state (PANAS, present state version).

The laboratory portion of the first session was intended to reduce anticipatory anxiety and acclimated participants to the rating scale routines. After the electrodes were attached, participants were shown a brief instructional video demonstrating how to make ratings using the mouse. They were then instructed to rest quietly for 10 min while psychophysiological data were collected. Next, participants made practice valence and arousal ratings to two neutral IAPS images not used in the experimental sessions. Finally, participants were administered a computer-generated PANAS.

Participants were scheduled to return to the laboratory approximately 3 days later and were randomly assigned an order of presentation of the two prime event conditions. Each subsequent laboratory session began with electrode attachment and the demonstration of the rating routine. Immediately after the baseline period, participants made valence and arousal ratings to the two neutral slides. Next, participants viewed the 10-min priming event, during which HR, SCL, and facial EMG were recorded. Directly after the 10-min priming event, participants made another valence and arousal rating. Participants were then shown the series of 24 IAPS photographs, with each image presented for 12 s followed by an intertrial interval of 30 s. We used the last 5 s of the intertrial interval as the pre-image baseline. A 2-s orienting phase was followed by a 10-s measurement interval. Immediately after the presentation of each IAPS image, participants made valence and arousal ratings.

Participants were then instructed to rest quietly during a 5-min recovery period. At this point, the PANAS was re-administered. Finally, participants engaged in an emotion expression task, included for pilot testing purposes and not reported here. Participants returned to the laboratory approxi-

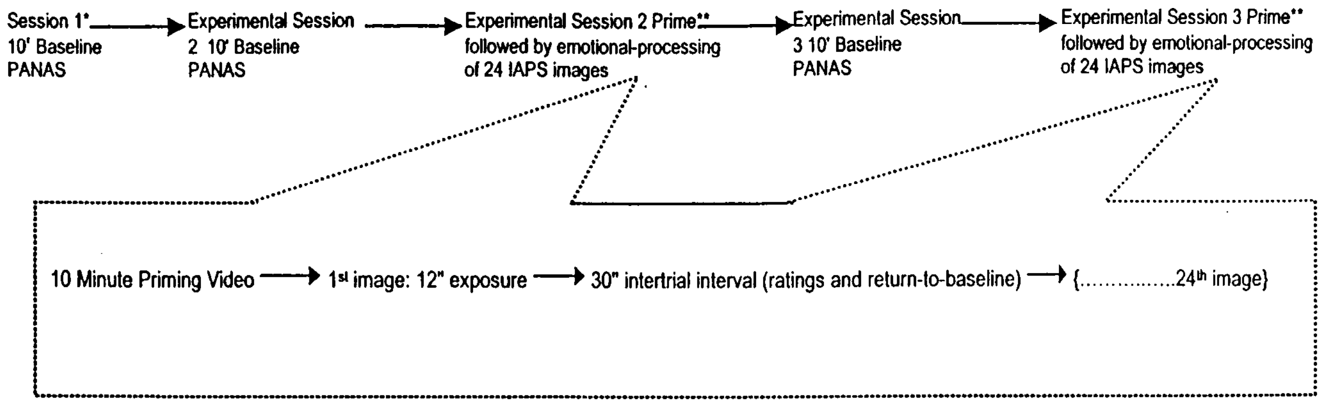

Figure 1. A schematic of the procedure. * Session 1 entailed a diagnostic interview followed by an initial assessment of baseline psychophysiology. ** The neutral and combat primes were presented in counterbalanced order. The two experimental sessions occurred approximately one week apart.

mately 1 week later and completed the experiment a second time under the other prime event condition.

Data Reduction and Analysis

Autonomic activity. All SCL data were log transformed (log10 [1 + value]), consistent with Lang et al. (1990). The baseline and prime periods were partitioned into 30-s segments, and a grand mean was calculated for HR and SCL on the basis of the mean for the 300 sample values from each individual segment within a period. A prime reactivity change score was derived for each prime event by subtracting the mean of the preceding baseline period from the mean for the total prime period. Autonomic responses were also evaluated during IAPS image processing. For each IAPS image, a change score was derived by subtracting the mean value for each index of autonomic activity for the 10 s of exposure (after the 2-s orienting interval) from the mean value from the pre-image baseline (5 s before image onset). Means of these change scores were then derived for the three IAPS categories (positive, neutral, and negative).

Facial EMG. For each IAPS image, a change score was derived by subtracting the mean EMG value from the 10 s of exposure (after the 2-s orienting interval) from the mean of the minimum (lowest) values from the pre-image baseline (measured every ½ s for the 5 s before image onset). Means of these change scores were then derived for the three IAPS categories (positive, neutral and negative).

In order to capture the relationship between reports of emotional experience and expressive–motor response, we plotted mean valence ratings by rank and EMG change scores, for all 24 IAPS images, within each of the prime event conditions, for the two study groups separately. We followed the guidelines of Lang et al. (1990), who examined the dimensional covariation of valence ratings and facial EMG normatively. Lang and his colleagues have shown a reliable quadratic association between mean-ranked valence ratings and zygomatic responses to IAPS images (Greenwald et al., 1989; Lang et al., 1990). There is a quadratic relationship between valence ratings and zygomatic activity because very negatively valenced images elicit a generalized EMG response because of the grimacing associated with disgust. These researchers have also shown a negative linear association between mean valence ratings and corrugator activity for IAPS images. Lang et al. (1993) argued that correspondence between valence ratings and facial–motor responses indexes individual differences in emotional expressivity. For example, relative to men, women show greater correspondence (correlations) normatively between zygomatic EMG and valence ratings (Lang et al., 1990). The analysis of dimensional covariation provides a summary index of the degree of integration of emotional responding across a range of stimuli. Individuals normatively manifest concordance or correspondence between their reports of emotional experience and expressive–motor behavior (e.g., stimuli experienced as highly pleasant elicit correspondingly high zygomatic EMG responses). In this study, each participant's valence ratings were ranked for all 24 images. Ties were reconciled by group mean ranks per prime event condition. Mean valence values per rank were then derived for each group per prime event condition.

Results

Participant Characteristics

Table 2 displays descriptive information on the two study groups. The PTSD group was significantly younger than the WAV group, $t(58) = 2.97, p < .01$, and less likely to be married (31.3% vs. 58.6%), $\chi^2(1, N = 61) = 4.62, p < .05$. The PTSD group had a greater mean CES score than the WAV group, $t(58) = 4.16, p < .001$. It should be emphasized, however, that mean scores for both groups reflect moderate to high exposure to potentially traumatizing war-zone events (Keane et al., 1989). The two groups did not differ on number of years of education or racial/ethnic identity. The two groups, as expected, were reliably different on all measures of psychopathology: The percentage of additional Axis-I diagnoses (for sake of brevity, only the four most frequent disorders are listed in Table 2), the BDI, $t(59) = 7.4, p < .0001$; the BAI, $t(59) = 7.1, p < .0001$; and the M–PTSD, $t(59) = 15.2, p < .0001$.

Baseline Activity

The baseline PANAS data were analyzed using a 2 (group) × 3 (session) × 2 (affectivity: positive vs. negative) repeated measures univariate analysis of variance (ANOVA). The only statistically significant effect to emerge from this analysis was an interaction of Group × Affectivity, $F(1, 102) = 11.14, p < .002$. The PTSD group reported reliably greater negative affectivity across all three sessions, relative to the WAV group. Contrary to a generalized restricted range of affect model, the reports of positive affectivity were not different between the two groups. The baseline mood of the PTSD group was consistent with anxious mood reported by individuals with anxiety disorders, in contrast with the anhedonia

Table 2
Descriptive Characteristics of the Two Study Groups

Variable	PTSD	WAV
Age*		
M	49.5	52.3
SD	2.8	5.5
No. of years of education		
M	14.62	15.97
SD	2.9	2.9
Race/ethnicity (%)		
Caucasian	81.3 (n = 26)	82.8 (n = 24)
African American	9.4 (n = 3)	13.8 (n = 4)
Hispanic or Latino	6.3 (n = 2)	3.4 (n = 1)
Axis-I diagnoses (% current; N = 29)		
Major depression	43.8	n/a
Dysthymia	15.6	n/a
Substance dependence	18.8	n/a
Social phobia	12.5	n/a
Combat Exposure Scale scores*		
M	29.3	19.2
SD	7.0	11.5
Mississippi Scale scores*		
M	124.0	64.5
SD	17.5	12.6
Beck Anxiety Inventory scores*		
M	20.8	2.9
SD	13.2	3.5
Beck Depression Inventory scores*		
M	24.9	4.9
SD	13.6	5.3

Note. N for the PTSD group is 32 and N for the WAV is 29, unless noted. Percentages that do not total 100 reflect missing data. PTSD = posttraumatic stress disorder; WAV = well-adjusted veterans.
* $p < .0001$.

and low positive affect associated with depression and mixed anxiety and depression (e.g., Watson, Clark, Weber, & Assenheimer, 1995).

Each baseline physiological variable was submitted to a 2 (group) × 3 (session) repeated measures ANOVA. There were no statistically reliable effects on any index of baseline physiological activity. These data suggest that there were no differential anticipatory arousal effects in our study. For sake of brevity, Table 3 displays descriptive data on mean PANAS, HR, SCL, corrugator EMG, and zygomatic major EMG activity during the initial baseline.

For all subsequent analyses, we covaried Combat Exposure scale scores, BDI scores, and negative affectivity scores. These variables were found to distinguish the PTSD and the WAV groups and may influence emotional behavior. Conceptually, by using these variables as covariates, we attempted to rule out the influence of differential reports of exposure to potentially traumatizing circumstances in the war zone, reports of symptoms of depression, and negative affectivity, on emotional processing in PTSD.

Priming Event Response

The postbaseline valence and arousal ratings were subtracted from the postpriming event ratings to index change in emotional response. These change scores were submitted to a 2 (group) × 2 (prime event) repeated measures analysis of covariance (ANCOVA). For valence ratings, there was a significant main effect of priming event, $F(1, 50) = 84.99$, $p < .0001$, which was qualified by a group of priming event interaction, $F(1, 50) = 8.98$, $p < .004$. The PTSD group reported more unpleasant feelings in response to the combat prime than the WAV group ($M = -3.3$, $SD = 2.3$ vs. $M = -2.37$, $SD = 1.3$, respectively); the valence change scores during the neutral prime were not different between the two groups (PTSD: $M = 0.35$, $SD = 1.8$; WAV: $M = -0.56$, $SD = 1.9$). The analysis of arousal ratings revealed a main effect of priming event, $F(1, 51) = 41.92$, $p < .0001$, and a main effect of group, $F(1, 51) = 3.9$, $p < .05$. Whereas each group reported greater arousal to the combat prime (PTSD: $M = 3.0$, $SD = 2.3$; WAV: $M = 1.9$, $SD = 2.6$) than the neutral prime (PTSD: $M = -0.17$, $SD = 2.3$; WAV: $M = -0.33$, $SD = 2.00$), the PTSD group experienced greater arousal overall.

The analysis of HR revealed differential arousal in the PTSD group, specifically to the combat prime event. The main effect of group approached statistical significance, $F(1, 57) = 3.83$, $p < .055$, and was qualified by an interaction of group and priming event, $F(1, 57) = 3.90$, $p < .05$. The PTSD group experienced greater mean HR change to the combat prime event than the WAV group (M = 2.4 BPM, SD = 2.9 vs. M = 0.29 BPM, SD = 2.4, respectively). The HR change scores to the neutral prime were not

Table 3
Baseline Mood Ratings and Physiological Activity

	PTSD			WAV		
Variable	*n*	*M*	*SD*	*n*	*M*	*SD*
First session baseline PANAS						
Positive affectivity	28	28.6	6.8	28	29.0	6.9
Negative affectivity	28	17.1	5.7	28	12.7	3.3
First session baseline physiological activity						
Heart rate (BPM)	32	68.9	10.1	23	67.6	15.1
Skin conductance level (μS)	29	1.70	1.5	23	2.37	3.3
Corrugator EMG (μV)	32	2.36	2.5	28	2.47	2.1
Zygomatic EMG (μV)	31	1.60	1.6	29	2.70	3.1

Note. Ns vary from missing data and out of range values. PTSD = posttraumatic stress disorder; WAV = well-adjusted veterans; PANAS = Positive and Negative Affect Schedule; BPM = beats per minute; EMG = electromyography.

statistically reliable (PTSD: $M = 0.68$ BPM, $SD = 3.65$; WAV: $M = 0.45$ BPM, $SD = 2.6$). The interaction effect was no longer reliable when each covariate was entered into the model (in any permutation). These results suggest that differential HR reactivity in the PTSD group relative to the WAV group is related not only to diagnostic status, but to continuous measures of combat exposure, depression, and negative affectivity as well. This is in part artifactual. When using an extreme groups approach in research with Vietnam combat veterans, each of the covariates are highly covaried with PTSD generally and with psychophysiological reactivity specifically (e.g., Orr, Claiborn, Altman, Forgue, & Pitman, 1990).

The analysis of mean SCL revealed no statistically significant effects. The untransformed mean SCL change scores for the neutral prime were as follows: PTSD: $M = 0.22$, $SD = .54$; WAV: $M = 0.35$, $SD = .78$; for the combat prime condition, the SCL change scores were as follows: PTSD: $M = 0.45$, $SD = 0.61$; WAV: $M = 0.35$, $SD = 0.63$.

The analysis of change from baseline corrugator EMG activity revealed a significant main effect of prime event, $F(1, 51) = 10.14$, $p < .002$, and no other reliable findings. Each group had greater expressive motor activity in the corrugator region to the combat prime, relative to baseline (PTSD: $M = 2.0$ μV, $SD = 5.7$; WAV: $M = 2.3$ μV, $SD = 2.8$), in comparison with neutral prime condition (PTSD: $M = 0.95$ μV, $SD = 1.4$; WAV: $M = 0.70$ μV, $SD = 1.1$). There were no statistically significant effects for EMG activity over the zygomatic region, for the neutral prime condition (PTSD: $M = 0.98$ μV, $SD = 3.1$; WAV: $M = 0.5$ μV, $SD = 0.72$), or the combat prime condition (PTSD: $M = 0.79$ μV, $SD = 0.90$; WAV: $M = 2.0$ μV, $SD = 6.3$).

Emotional Processing of IAPS Images

Self-report of emotional valence and arousal. The mean valence and arousal ratings, in each Prime Condition × Emotional Valence Category, are presented in Table 4. These data were submitted to a 2 (group) × 2 (prime event) × 3 (emotional valence category) repeated measures ANCOVA. For valence ratings, this analysis revealed a main effect of emotional valence category, $F(2, 102) = 20.52$, $p < .0001$, which provided ostensibly a manipulation check for the IAPS slides chosen for the study. All of the participants reported more pleasant reactions to positive images, in comparison with the neutral (Fisher's least significant difference [LSD], $p < .001$), and the negative images (Fisher's LSD, $p < .001$). In addition, all of the participants responded more positively to the neutral IAPS images, in comparison with the negative stimuli, (Fisher's LSD, $p < .001$).

The analysis of valence ratings also yielded a significant Group × Prime Event × Emotional Valence Category interaction, $F(2, 102) = 3.02$, $p < .05$. The analysis of simple interaction effects of Group × Prime Condition, within each valence category revealed a significant effect for neutral images only. The PTSD group reported the neutral images as less pleasant, under the combat prime condition, relative to the WAV group, $F(1, 52) = 5.6$, $p = .02$. A simple main effect analysis of prime condition, within group and valence category, revealed two significant effects in the PTSD group only. The PTSD group rated positive and neutral images as less pleasant in the combat prime condition relative to the neutral prime condition, $F(1, 52) = 11.6$, $p < .001$; $F(1, 52) = 8.3$, $p < .006$, respectively.

Table 4
Emotional Processing of IAPS Images by Category

	PTSD									WAV								
	Positive			Neutral			Negative			Positive			Neutral			Negative		
Variable	*n*	*M*	*SD*	*n*	*M*	*SD*	*n*	*M*	*SD*	*n*	*M*	*SD*	*n*	*M*	*SD*	*n*	*M*	*SD*
Valence ratings																		
Neutral prime	30	6.8	1.3	30	4.8	.59	30	2.5	.93	28	6.9	0.83	28	4.9	0.33	28	2.8	.97
Combat prime	30	6.4	1.6	30	4.4	1.0	30	2.7	.47	28	6.7	0.95	28	5.0	0.26	28	2.9	.89
Arousal ratings																		
Neutral prime	31	5.4	1.3	31	4.7	1.2	31	5.5	1.3	29	5.1	1.7	29	4.3	1.4	29	4.9	1.4
Combat prime	31	5.1	1.5	31	4.6	1.4	31	5.5	1.4	29	5.0	1.5	29	4.4	1.3	29	4.9	1.4
Zygomatic EMG (μV)																		
Neutral prime	30	1.2	1.5	30	0.78	.85	30	0.77	.98	29	1.1	1.5	29	0.79	0.95	29	0.64	0.58
Combat prime	30	0.70	0.60	30	0.64	.77	30	0.63	.81	29	1.3	1.6	29	0.96	0.81	29	1.0	1.9
Corrugator EMG (μV)																		
Neutral prime	32	0.15	0.64	32	0.67	.67	32	0.99	1.4	28	0.34	1.4	28	0.80	1.2	28	1.1	1.3
Combat prime	32	0.50	2.6	32	1.3	3.1	32	1.8	3.7	28	0.40	0.58	28	1.0	0.87	28	1.3	1.3
Skin conductance level (μS, untransformed)																		
Neutral prime	31	0.06	0.10	31	0.08	0.10	31	0.09	0.10	29	0.10	0.19	29	0.15	0.25	29	0.16	0.31
Combat prime	32	0.07	0.15	32	0.06	0.07	32	0.07	0.14	28	0.12	0.20	28	0.11	0.19	28	0.11	0.19
Heart rate (BPM)																		
Neutral prime	29	4.9	2.3	29	4.7	1.9	29	4.6	2.6	27	4.3	2.2	27	4.1	2.2	27	3.9	2.4
Combat prime	29	4.4	2.1	29	4.5	2.5	29	4.4	1.9	27	4.7	2.3	27	4.4	2.8	27	3.7	2.7

Note. *ns* vary from missing data and out of range values. Valence and arousal values, unlike the physiological measures, are not change scores. IAPS = International Affective Picture System; PTSD = posttraumatic stress disorder; WAV = well-adjusted veterans; EMG = electromyography; BPM = beats per minute.

A similar statistical analysis of arousal ratings revealed only a main effect of emotional valence category, $F(2, 106) = 5.7, p < .006$, which also provided a manipulation check for the IAPS images chosen for the study. Pairwise comparisons collapsed across prime event and group revealed that all of the participants reported less arousal to the neutral IAPS images, in comparison with the positive stimuli (Fisher's LSD, $p < .0001$), and the negative images (Fisher's LSD, $p < .0001$). The difference between the arousal ratings for positive images and negative images was not statistically significant.

Peripheral autonomic responses. The mean SCL and HR change scores are presented in Table 4. The analysis of skin conductance change scores revealed a significant Prime × Valence interaction, $F(2, 102) = 3.5, p < .04$, and no other statistically significant effects. In the neutral prime condition, exclusively, participants had lower SCL change scores to positive images, in comparison to neutral (Fisher's LSD, $p < .02$), and negative (Fisher's LSD, $p < .04$) images.

The analysis of HR change scores during IAPS image processing revealed a significant main effect of group, $F(1, 47) = 3.97$, $p < .05$, and no other statistically reliable effects. Participants in the PTSD group had higher heart rates while processing all images, in comparison with individuals in the WAV group.

Facial EMG. The mean zygomatic and corrugator EMG change scores are also presented in Table 4. Each EMG measure was submitted to a 2 (group) × 2 (prime event) × 3 (emotional valence category) repeated measures ANCOVA. The analysis of zygomatic EMG change scores revealed a main effect of emotional valence category, $F(2, 102) = 3.4, p < .04$. Pairwise comparisons, collapsed across group and prime event, revealed that all participants had larger zygomatic change scores to positive images, in comparison with the neutral (Fisher's LSD, $p < .05$), and negative (Fisher's LSD, $p < .05$) images. There were no reliable differences in zygomatic change scores between neutral and negative IAPS images.

The analysis of zygomatic EMG change scores also revealed a significant Group × Prime Event × Valence category interaction, $F(2, 102) = 3.8, p < .03$. The analysis of simple interaction effects of Group × Prime Condition, within each valence category, revealed a significant effect for positively valenced images only. The PTSD group had less expressive motor activity in the zygomatic region to the positive IAPS images, under the combat prime condition, relative to the WAV group, $F(1, 52) = 4.4, p = .04$. A simple main effect analysis further revealed that the PTSD group had lower mean zygomatic EMG responses to the positive images in the combat prime condition, relative to the neutral prime condition, $F(1, 52) = 3.98, p < .05$.

In contrast, the overall analysis of corrugator EMG change scores revealed only a significant main effect of emotional valence category, $F(2, 102) = 35.2, p < .0001$. Pairwise comparisons collapsed across prime event and group revealed that all of the participants had larger corrugator change scores to negative images, in comparison with the neutral (Fisher's LSD, $p < .001$) and positive (Fisher's LSD, $p < .001$) images. In addition, all of the participants responded to the positive IAPS images, relative to the neutral images, with reliably less corrugator activity (Fisher's LSD, $p < .001$).

Dimensional covariation of EMG and valence ratings. Figures 2–5 show scatterplots and best fit quadratic regression curves, depicting the relationship between mean valence ratings by mean rank and zygomatic change scores, for all 24 IAPS images for the two groups and the two prime event conditions separately. Each set of values was submitted to a bivariate, quadratic curve-fitting regression procedure. For the WAV group, the quadratic correla-

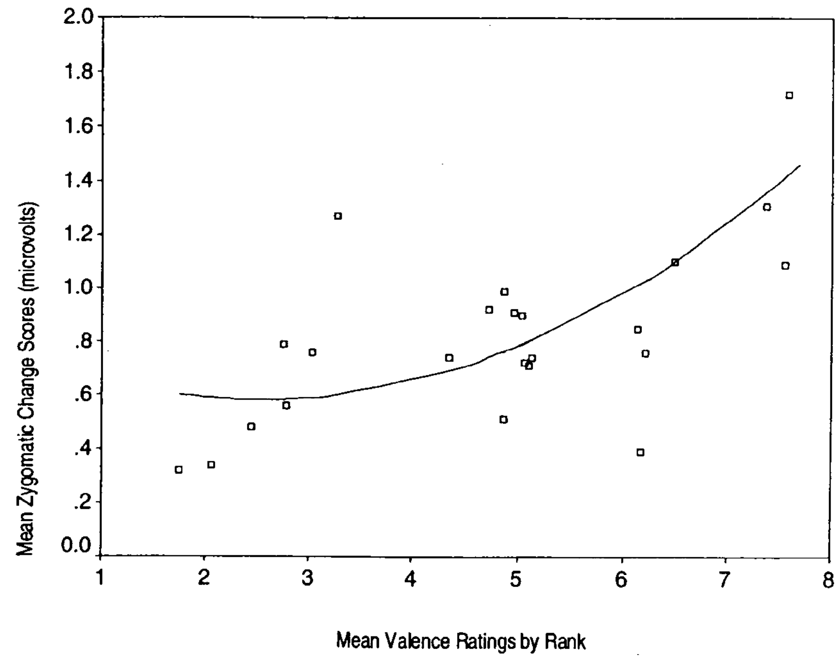

Figure 2. Dimensional covariation between mean valence ratings by rank and zygomatic change scores for all 24 images: well-adjusted veterans (WAV) group, neutral prime condition ($r = .51$), $F(21) = 10.92, p < .001$.

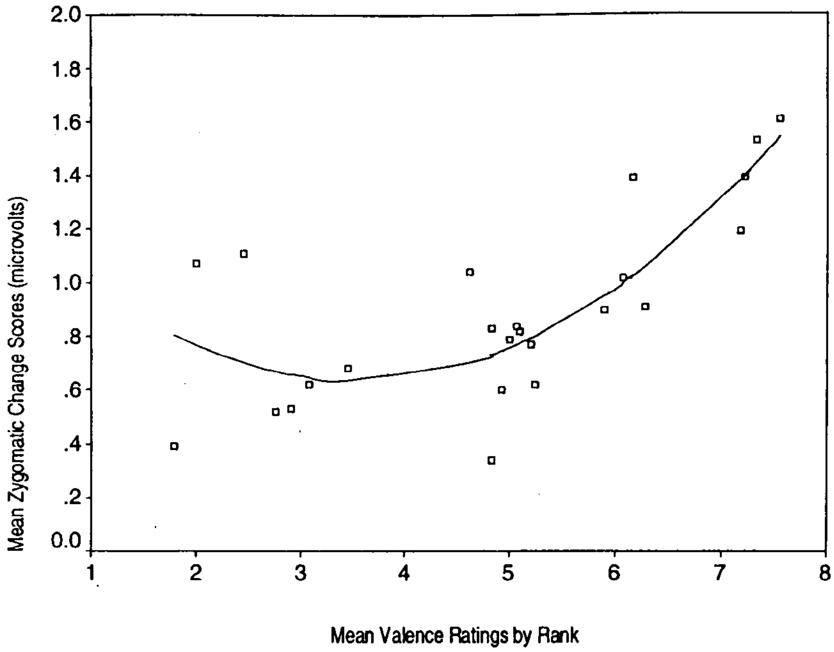

Figure 3. Dimensional covariation between mean valence ratings by rank and zygomatic change scores for all 24 images: well-adjusted veterans (WAV) group, combat prime condition ($r = .63$), $F(21) = 18.07$, $p < .0001$.

tion between mean valence and zygomatic change scores in the neutral prime event condition was .51, $F(21) = 10.92$, $p < .001$, and .63 in the combat prime event condition, $F(21) = 18.07$, $p < .0001$. The corresponding quadratic correlation for the PTSD group, in the neutral prime event condition, was .26, $F(21) = 3.74$, $p < .04$, and .11 in the combat prime event condition, $F(21) = 1.3$,

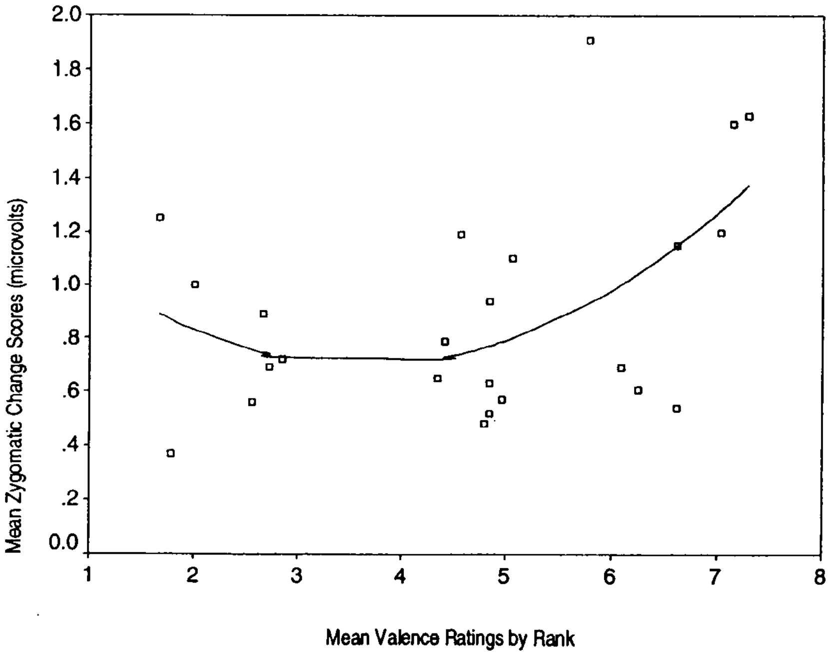

Figure 4. Dimensional covariation between mean valence ratings by rank and zygomatic change scores for all 24 images: posttraumatic stress disorder (PTSD) group, neutral prime condition ($r = .26$), $F(21) = 3.74$, $p < .04$.

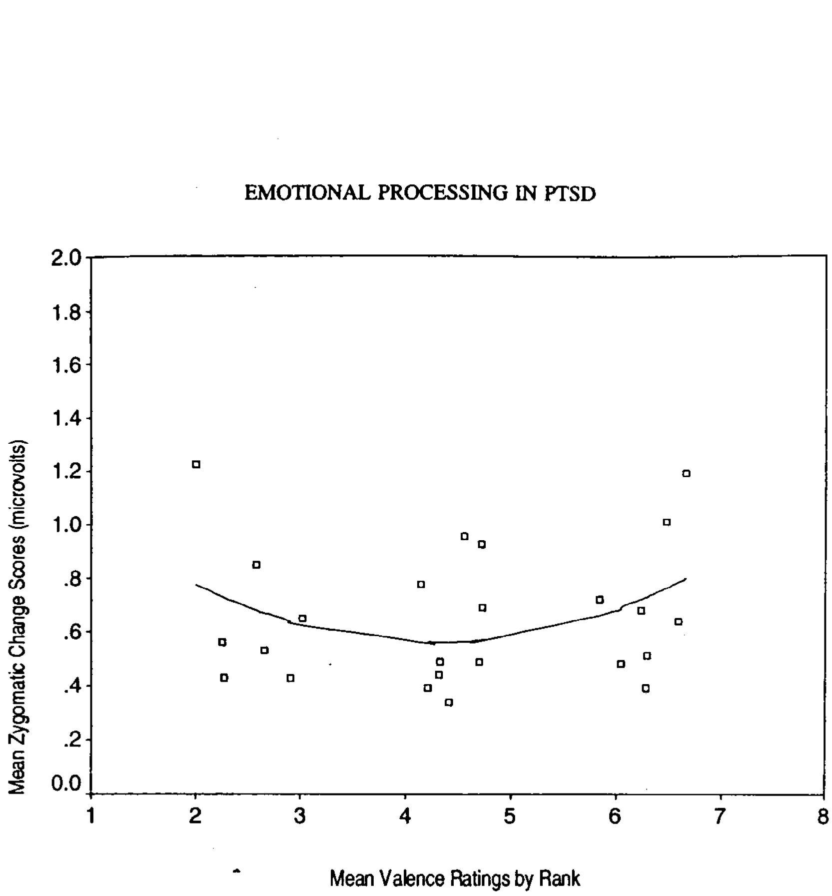

Figure 5. Dimensional covariation between mean valence ratings by rank and zygomatic change scores for all 24 images: posttraumatic stress disorder (PTSD) group, combat prime condition ($r = .11$), $F(21) = 1.35, p < .28$.

$p < .28$). A Z test was used to examine differences in correlation coefficients between the two groups for the two prime event conditions. The PTSD group showed a range of facial EMG responses that reliably corresponded to the respective valence ratings under the neutral prime condition. However, the PTSD group's facial expressive–motor displays to the range of IAPS images rated from pleasant to unpleasant were uncorrelated and reliably different from the WAV group in the combat prime condition exclusively, $Z = 2.3, p < .01$.

We also examined the bivariate linear best fit regression line between mean valence ratings by mean rank and corrugator EMG change scores, for the two groups and the two prime event conditions separately. For the WAV group, the linear correlation between mean valence and corrugator change scores in the neutral prime event condition was .30, $F(22) = 8.5, p < .008$, and .57 in the combat prime event condition, $F(22) = 28.8, p < .0001$. The corresponding linear correlation for the PTSD group, in the neutral prime event condition, was .55, $F(22) = 26.3, p < .0001$, and .39 in the combat prime event condition, $F(22) = 13.8, p < .001$. There were no statistically reliable differences between these correlations, suggesting that the groups demonstrated equivalent concordance between expressed negative affect and ratings of pleasantness.

Discussion

In this study, we examined the emotional-processing consequences of exposure to trauma cues in PTSD patients and combat veterans free from psychiatric disorder. The results were inconsistent with the generally accepted concept of emotional numbing in PTSD. No facet of our findings suggests a generalized restricted range of emotional experience or expression of emotion specific to PTSD. After exposure to an emotionally neutral priming event, both groups responded comparably and distinctly to the three categories of emotionally valenced stimuli (positive, neutral, and negative), assessed by self-report, peripheral autonomic responses, and expressive–motor activity. There was also no evidence of a generalized suppression of emotional response in individuals with PTSD after exposure to a trauma-related challenge. The results, however, were consistent with the concept that emotional-processing deficits in PTSD are secondary, at least in part, to reactions to trauma reminders. The PTSD patients, in contrast with control participants, were less responsive to positively valenced emotional stimuli only after they were exposed to a trauma-related priming challenge.

The locus of the emotional-processing deficit that distinguished the PTSD group from the WAV group was chiefly in facial expressive–motor behavior. This result suggests that emotional deficits in PTSD might be best characterized as a trauma-cue, response-dependent diminution in expressive behavior to emotionally pleasant stimuli. Although the PTSD group reported reductions in valence reactions to positive images after being exposed to the combat prime, they could be distinguished from the WAV group only by reduced expressive behavior that would otherwise signal positive affect. This effect could not be explained by the PTSD group exhibiting facial displays associated with aversive reactions to the putatively positive images, as a function of a trauma-primed state. Participants with and without PTSD reported comparable arousal ratings, corrugator EMG responses, and SCL responses to the positively valenced images, in both prime-event conditions. In addition, corrugator EMG and valence ratings were linearly associated in both groups, regardless of prime condition. However, as can be seen graphically in Figure 5, there appears to be a decoupling of judgments of emotional experience and facial–expressive motor reactions as a consequence of exposure to trauma-related cues in PTSD.

There are several potential explanations for the finding that trauma priming is associated with diminished facial expressions of positive, appetitive emotion in PTSD. It could be that report of feeling and facial–motor responses are decoupled, as shown in Figure 5, because participants with PTSD are susceptible to the biasing effects of social desirability in their ratings of emotional response. That is, patients with PTSD may have rated the IAPS images in regard to what they should feel or what others might feel. This seems unlikely given the general negative self-report bias seen in veterans with PTSD (e.g., Fairbank, McCaffrey, & Keane, 1985). Alternatively, when patients with PTSD are exposed to a trauma context, they may require more intensive and sustained stimulation to trigger expressive behavior consistent with positive emotion. It could be that the threshold is increased for facial–motor reactions inconsistent with trauma-primed emotional states in PTSD (cf. Cacioppo et al., 1992). In PTSD, the raised threshold for the expressive–motor aspect of positive emotion is likely to be the result of transitory drains in emotional-processing capacity and reduced accessibility of cognitive structures associated with pleasant feelings (Litz, 1992). Exposure to trauma cues may drain processing resources to a sufficient degree so that deep processing of affective information inconsistent with a negative mood is impeded. Lang (1985) posited that individuals who report feeling, but do not manifest a somatic response consistent with the experiential component of feeling, are failing to access deeper, less accessible aspects of an emotion network. It could be that participants with PTSD, even after exposure to a trauma prime, were able to process the positive images to a sufficient degree allowing for an appraisal of stimulus content. The trauma priming, however, reduced patients' capacity to access fully the positive emotional network, which when activated in quiescent states elicits full, synchronous emotional reactions.

The reduced facial expression of positive emotion in PTSD, specific to a trauma-cue context, is likely to be the result of automatic cognitive and emotional processes, given that EMG responses have been shown to be relatively free from the influences of strategy or intention (e.g., Cacioppo, Bush, & Tassinary, 1992). Patients with PTSD may be subject, unwittingly, to suppressed expressive reactions to positive stimuli while attempting to cope with their responses to traumatic contexts. Top-down executive cognitive processes activated by trauma cues may also lead to reduced expressive behavior to positive stimuli. It might be that the display rules that moderate expressive behavior are particularly conservative when patients with PTSD are coping with conditioned aversive emotional responses to trauma-related cues (Ansorge, Litz, & Orsillo, 1996).

We also expected the two groups to be distinct in their pleasantness ratings of positive IAPS images specifically after the combat prime. Instead, the PTSD group was distinguished from the WAV group by their rating of the neutral images as less pleasant in the combat prime condition. This unexpected result suggests that appraisal of hedonically neutral stimuli may be altered in trauma-related contexts in PTSD. A cursory examination of the images used in this study suggests that many of the neutral images have less information value than the positive and negative photographs. For example, several neutral images are human male faces with little or no contextual cues in the background or foreground that could be used to construct a clear idea of what is taking place. The neutral images may have been more ambiguous and, thus, more subject to interpretation and appraisal consistent with a trauma-cue context. However, the PTSD group's altered experience of pleasantness to neutral images did not translate into reduced facial expressive–motor behavior to these stimuli, relative to the control group. Nevertheless, it may be fruitful in future research to examine the mediating influences of not only the degree of exposure to trauma-related contexts and stimulus valence on emotional-processing capacity in PTSD, but the degree of ambiguity and uncertainty as well (see Duncan & Fiske, 1985). Finally, an ideographic trauma-priming procedure (e.g., imaginal exposure to a personalized script of a specific traumatic event; Pitman et al., 1987) may induce the expected group differences in emotional experience to positively valenced stimuli.

Another unexpected finding was that the PTSD group was also distinguished from the WAV group by having greater heart rates to all emotional stimuli, regardless of prime context. This result suggests that individuals with PTSD were experiencing each trial as demanding. Although unanticipated, this finding makes sense in light of the stimulus presentation routine. The emotional content of each IAPS trial could not have been anticipated by participants during the long intertrial interval, which may have heightened arousal reactions, regardless of the valence of the stimulus or the prime context. It may be the case that when individuals with PTSD are exposed to unpredictable, uncertain emotional stimulation, they prepare automatically for threat or demand. This interpretation is supported by conceptual models that emphasize the central role of unpredictability and uncontrollability in the etiology and maintenance of PTSD (Foa et al., 1992). The generalized physiological arousal of the PTSD group must be considered relative to their normal emotional responses in the neutral prime condition and their attenuated expressive behavior to positive stimuli following the trauma-relevant prime. This pattern of findings suggests that PTSD is associated with vigilant readiness to respond to threat in a context that promotes uncertainty, but this state does not appear to preclude the capacity for modulated emotional responses in a neutral context or for reduced expressive behavior in relation to positive stimuli that occur in a trauma-related context.

Although the PTSD group showed greater responsiveness to the combat prime (in particular, increased unpleasant emotion), they were consequently not more (or less) reactive to negatively valenced emotional stimuli, in comparison with the WAV group. This null result suggests that exposure to a trauma-related context does not necessarily augment subsequent emotional behavior to hedonically negative stimuli. Associative network models of cognition and emotion in PTSD would predict that a trauma prime would lead to a cascading, spreading activation process, influencing emotional behavior in a mood-consistent manner. It might be that this type of negative affect augmentation is likely to emerge only when patients with PTSD are processing more explicitly self-referential material (McNally et al., 1994). In this respect, there remains the possibility that a more potent trauma-related priming challenge may have elicited greater emotional responses to negatively valenced stimuli. As suggested above, researchers should consider using an ideographic, trauma-script priming procedure, which may elicit more intense negatively valenced emotional responses in participants with PTSD. If our results are replicated, it might be considered that the consequence of spreading activation of the trauma network in PTSD is reduced accessibility of (full) positive emotion network activation and increased accessibility of threat-related emotional processing (e.g., Litz et al., 1996), but no greater non-threat-related negative affective responsivity.

The results of this study suggest that the symptom class of emotional numbing in PTSD requires reevaluation. When the emotional-processing characteristics of the negative symptoms associated with other psychiatric disorders have been systematically evaluated, a similar conclusion has ensued. Kring and her colleagues found a decoupling of emotional experience and expression in schizophrenia. In terms of the report of emotional feeling, schizophrenic patients were as responsive to positive and negative emotion-eliciting stimuli as were matched control participants; however, they were unresponsive in regard to facial expressions of both positive and negative emotion (e.g., Kring & Neale, 1996). Thus, when examined empirically, *flat affect* in schizophrenia appears to be a generalized expressive–motor deficit, rather than a generalized incapacity to experience feeling. Several researchers have examined the emotional-processing correlates of anhedonia, which entails difficulty with feeling pleasure and is a characteristic found in affective disorders and individuals at risk for schizophrenia-spectrum disorders. For example, Fitzgibbons and Simon (1992) used a methodology very similar to the one used in this study to examine emotional processing in anhedonics. Anhedonics in their study had lower valence ratings to positive, neutral, and negative IAPS slides, but contrary to expectations, they had greater zygomatic reactions to positive images. This finding, in light of our results, suggests that emotional processing in PTSD is distinct from anhedonia, which entails indiscriminant negatively biased ratings of feeling, with no concomitant changes in expressive behavior.

What might be the consequences of a facial expressive deficit specific to positive stimuli, secondary to trauma contexts in PTSD? Given that emotion is considered a disposition to respond to the environment in an adaptive, relational manner, the reduction in expressive–motor responses consistent with pleasant feeling is likely to have maladaptive effects. Patients with PTSD are likely to be handicapped in their capacity to display positive feelings as a general characteristic, especially if they have been recently traumatized or are frequently exposed to reminders. Over time, the patient with PTSD may require an extended period free from reexperiencing their trauma to exhibit a broad range of positively valenced feeling. They will likely need to be in a very familiar and safe context to manifest a variety of premorbid emotional repertoires related to positive emotion. Significant others may also need to provide more intense, sustained, and unambiguously affiliative responses in order to stimulate discernable positive emotional behaviors in traumatized individuals. However, because social contexts are often ambiguous and uncertain and significant others may have difficulty meeting the emotional needs of traumatized individuals (Tarrier, 1996), patients with PTSD are likely to have difficulty sustaining sufficient processing resources in order to express positive feeling. This process may lead to interpersonal disengagement and estrangement.

Future research should examine more sustained emotional demands, in and outside the laboratory in PTSD. Challenge tasks could entail a variety of emotionally evocative materials (film clips, live interpersonal exchanges, etc.). It will also be revealing to manipulate various aspects of trauma-cue processing, in order to examine the necessary and sufficient features that lead to subsequent emotional-processing deficits. This is particularly important given that it remains unclear from this study whether the trauma prime induced a generic negative mood or actually triggered ideographic trauma memories. A more stringent test of emotional-processing deficits specific to trauma-cue contexts in PTSD would be to use a generic aversive emotion challenge (e.g., a video depicting horrific content) to both patients and control participants. In future studies, anxious arousal could be also examined independent of trauma-related content by administering sympathetic nervous system activators, such as yohimbine (e.g., Morgan et al., 1995). Finally, although the principal results of this study could not be accounted for by reports of symptoms of depression, future research should nevertheless use a control group of depressed individuals who have a history of exposure to potentially traumatizing events (and no current PTSD). Given the experiential overlap between symptoms of depression and emotional processing in PTSD, this evaluation seems especially crucial.

A few speculations about the clinical relevance of our findings is warranted. Our results suggest that there may be a primacy to conditioned emotional reactions in PTSD. Although our data suggest that emotional stimuli of any kind, regardless of context, may elicit a readiness to respond defensively, PTSD patients are nevertheless capable of integrated modulation of emotion. When exposed to reminders of their trauma, PTSD patients appear to be less expressive of positive feeling. The implication is that if conditioned emotional reactions to trauma cues are reduced or eliminated through extinction procedures such as exposure therapy, emotional-processing deficits should abate.

References

American Psychiatric Association. (1994). *Diagnostic and statistical manual of mental disorders (4th ed.).* Washington, DC: Author.

Ansorge, S., Litz, B. T., & Orsillo, S. (1996). The role of meta-mood in post-traumatic stress disorder. *PTSD Clinical Quarterly, 6,* 38–41.

Beck, A. T., Epstein, N., Brown, G., & Steer, R. (1988). An inventory for measuring clinical anxiety: Psychometric properties. *Journal of Consulting and Clinical Psychology, 56,* 893–897.

Beck, A. T., Ward, C., Mendelsohn, H., Mock, J., & Erbaugh, J. (1961). An inventory for measuring depression. *Archives of General Psychiatry, 4,* 561–571.

Blake, D. D., Weathers, F. W., Nagy, L. N., Kaloupek, D. G., Klauminzer, G., Charney, D. S., & Keane, T. M. (1990). A clinician rating scale for assessing current and lifetime PTSD: The CAPS-1. *Behavior Therapist, 13,* 187–188.

Bradley, M., Greenwald, M., Petry, M., & Lang, P. (1992). Remembering pictures: Pleasure and arousal in memory. *Journal of Experimental Psychology: Learning, Memory and Cognition, 18,* 379–390.

Cacioppo, J. T., Bush, L. K., & Tassinary, L. G. (1992). Microexpressive facial actions as a function of affective stimuli: Replication and extension. *Personality and Social Psychology Bulletin, 18,* 515–526.

Cacioppo, J. T., Uchino, B. N., Crites, S. L., Snydersmith, M. A., Smith, G., Bernston, G. G., & Lang, P. J. (1992). Relationship between facial expressiveness and sympathetic activation in emotion: A critical review, with emphasis on modeling underlying mechanisms and individual differences. *Journal of Personality and Social Psychology, 62,* 110–128.

Center for the Study of Emotion and Attention, National Institute of Mental Health. (1999). *The International Affective Picture System: Digitized photographs.* Gainesville: University of Florida, Center for Research in Psychophysiology.

Chemtob, C., Roitblat, H., Hamada, R., Carlson, J., & Twentyman, C. (1988). A cognitive–action theory of post-traumatic stress disorder. *Journal of Anxiety Disorders, 2,* 253–275.

Davidson, J., & Foa, E. (1991). Diagnostic issues in posttraumatic stress disorder: Considerations for the *DSM–IV. Journal of Abnormal Psychology, 100,* 346–355.

Duncan, S. D., & Fiske, D. W. (1985). *Interaction, structure, and strategy.* Cambridge, England: Cambridge University Press.

Fairbank, J. A., McCaffrey, R. J., & Keane, T. M. (1985). Psychometric detection of fabricated symptoms of posttraumatic stress disorder. *American Journal of Psychiatry, 142,* 501–503.

Fitzgibbons, L., & Simons, R. F. (1992). Affective response to color–slide stimuli in subjects with physical anhedonia: A three-systems analysis. *Psychophysiology, 29,* 613–620.

Foa, E., Riggs, D., & Gershuny, B. (1995). Arousal, numbing, and intrusion: Symptom structure of PTSD following assault. *American Journal of Psychiatry, 152,* 116–120.

Foa, E. B., Steketee, G., & Rothbaum, B. O. (1989). Behavioral/cognitive conceptualizations of post-traumatic stress disorder. *Behavior Therapy, 20,* 155–176.

Foa, E., Zinbarg, R., & Rothbaum, B. (1992). Uncontrollability and unpredictability in posttraumatic stress disorder: An animal model. *Psychological Bulletin, 112,* 218–238.

Fowles, D. C., Christie, M. J., Edelberg, R., Grings, W. W., Lykken, D. T., & Venables, P. H. (1981). Publication recommendations for electrodermal research. *Psychophysiology, 18,* 232–239.

Fridlund, A., & Cacioppo, J. (1986). Guidelines for human electromyographic research. *Psychophysiology, 23,* 567–589.

Greenwald, M. K., Cook, E. W. III, & Lang, P. J. (1989). Affective judgment and psychophysiological response: Dimensional covariation in the evaluation of pictorial stimuli. *Journal of Psychophysiology, 3,* 51–64.

Horowitz, M. (1986). *Stress response syndromes* (2nd ed.). Northvale, NJ: Jason Aronson.

Joseph, S., Yule, W., & Williams, R. (1995). Emotional processing in survivors of the Jupiter cruise ship disaster. *Behaviour Research and Therapy, 33,* 187–192.

Keane, T. M., Caddell, J. M., & Taylor, K. L. (1988). Mississippi Scale for Combat-Related PTSD: Three studies in reliability and validity. *Journal of Consulting and Clinical Psychology, 56,* 85–90.

Keane, T. M., Fairbank, J. A., Caddell, J. M., Zimering, R. T., Taylor, K. L., & Mora, C. A. (1989). Clinical evaluation of a measure to assess combat exposure. *Psychological Assessment: A Journal of Consulting and Clinical Psychology, 1,* 53–55.

Keane, T. M., Fairbank, J. A., Caddell, J. M., Zimering, R. T., & Bender, M. E. (1985). A behavioral approach to assessing and treating post-traumatic stress disorder in Vietnam veterans. In C. R. Figley (Ed.), *Trauma and its wake* (pp. 257–294). New York: Brunner/Mazel.

Kring, A. M., & Neale, J. M. (1996). Do schizophrenic patients show a disjunctive relationship among expressive, experiential, and psychophysiological components of emotion? *Journal of Abnormal Psychology, 105,* 249–257.

Lang, P. J. (1985). The cognitive psychophysiology of emotion: Fear and anxiety. In A. H. Tuma & J. Maser (Eds.), *Anxiety and the anxiety disorders* (pp. 131–170). Hillsdale, NJ: Erlbaum.

Lang, P., Bradley, M., & Cuthbert, B. (1999). *International Affective Picture System (IAPS): Technical manual and affective ratings.* Gainesville: University of Florida, Center for Research in Psychophysiology.

Lang, P. J., Greenwald, M. K., Bradley, M. M., & Hamm, A. O. (1993). Looking at pictures: Affective, facial, visceral, and behavioral reactions. *Psychophysiology, 30,* 261–273.

Leventhal, H. (1984). A perceptual–motor theory of emotion. In K. R. Scherer & P. Ekman (Eds.), *Approaches to emotion* (pp. 271–291). New York: Academic Press.

Litz, B. (1992). Emotional numbing in combat-related post-traumatic stress disorder: A critical review and reformulation. *Clinical Psychology Review, 12,* 417–432.

Litz, B. T., & Keane, T. M. (1989). Information-processing in anxiety disorders: Application to the understanding of post-traumatic stress disorder. *Clinical Psychology Review, 9,* 243–257.

Litz, B. T., Schlenger, W. E., Weathers, F. W., Cadell, J. M., Fairbank, J. A., & LaVange, L. M. (1997). Predictors of emotional numbing in posttraumatic stress disorder. *Journal of Traumatic Stress, 10,* 607–617.

Litz, B. T., Weathers, F. W., Monaco, V., Herman, D. S., Wulfsohn, M., Marx, B., & Keane, T. M. (1996). Attention, arousal and memory in post-traumatic stress disorder. *Journal of Traumatic Stress, 9,* 497–519.

Malloy, P. F., Fairbank, J. A., & Keane, T. M. (1983). Validation of a multimethod assessment of posttraumatic stress disorders in Vietnam veterans. *Journal of Consulting and Clinical Psychology, 51,* 488–494.

McNally, R. J., Litz, B. T., Prassas, A., Shin, L. M., & Weathers, F. W. (1994). Emotional priming of autobiographical memory in post-traumatic stress disorder. *Cognition and Emotion, 8,* 351–367.

Morgan, C. A., Grillon, C., Southwick, S., Nagy, L. M., Davis, M., Krystal, J. H., & Charney, D. S. (1995). Yohimbine facilitated acoustic startle in combat veterans with post-traumatic stress disorder. *Psychopharmacology, 117,* 466–471.

Orr, S., Claiborn, J., Altman, B., Forgue, D., & Pitman, R. (1990). Psychometric profile of posttraumatic stress disorder, anxious, and healthy Vietnam veterans: Correlations with psychophysiologic responses. *Journal of Consulting and Clinical Psychology, 58,* 329–335.

Pallmeyer, T., Blanchard, E., & Kolb, L. (1986). The psychophysiology of combat induced post-traumatic stress disorder in Vietnam veterans. *Behavior Research and Therapy, 24,* 645–652.

Pitman, R. K., Orr, S. P., Forgue, D. F., de Jong, J. B., & Claiborn, J. M. (1987). Psychophysiologic assessment of posttraumatic stress disorder imagery in Vietnam combat veterans. *Archives of General Psychiatry, 44,* 970–975.

Pitman, R. K., van der Kolk, B. A., Orr, S. P., & Greenberg, M. S. (1990). Naloxone reversible analgesic response to combat-related stimuli in post-traumatic stress disorder. *Archives of General Psychiatry, 47,* 541–544.

Rauch, S. L., van der Kolk, B. A., Fisler, R. E., & Alpert, N. M. (1996). A symptom provocation study of posttraumatic stress disorder using positron emission tomography and script-driven imagery. *Archives of General Psychiatry, 53,* 380–387.

Spitzer, R., Williams, J., Gibbons, M., & Faust, M. (1989). *Structured Clinical Interview for* DSM–III–R–*Non-patient edition (SCID-NP).* New York: New York State Psychiatric Institute, Biometrics Research Department.

Tarrier, N. (1996). An application of expressed emotion to the study of PTSD: Preliminary findings. *Clinical Psychology and Psychotherapy: An International Journal of Theory and Practice, 3,* 220–229.

van der Kolk, B. (1987). *Psychological trauma.* Washington, DC: American Psychiatric Press.

van der Kolk, B., Greenberg, M., Boyd, H., & Krystal, J. (1985). Inescapable shock, neurotransmitters, and addiction to trauma: Toward a psychobiology of post-traumatic stress. *Biological Psychiatry, 20,* 314–325.

Watson, D., Clark, L. A., & Tellegen, A. (1988). Development and validation of brief measures of positive and negative affect: The PANAS scales. *Journal of Personality and Social Psychology, 54,* 1063–1070.

Watson, D., Clark, L., Weber, K., & Assenheimer, J. (1995). Testing a tripartite model: II. Exploring the symptom structure of anxiety and depression in student, adult, and patient samples. *Journal of Abnormal Psychology, 104,* 15–25.

Weathers, F. W., Herman, D. S., Litz, B. T., McNally, R. J., Meron, A., Palmieri, P., & Keane, T. M. (1996, November). *Selective-processing of trauma-relevant stimuli in war-zone-related posttraumatic stress disorder.* Paper presented at the annual meeting of the Association for Advancement of Behavior Therapy, New York.

Weathers, F. W., & Litz, B. T. (1994). Psychometric properties of the clinician administered PTSD scale. *PTSD Research Quarterly, 5,* 2–6.

Received February 5, 1998
Revision received January 26, 1999
Accepted May 4, 1999 ■